Exhibit 10

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Specific Causation Expert Report: Allan Wayne Howard

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VII. Kidney Cancer Risk Associated with TCE

The International Agency for Research on Cancer (IARC) classifies Trichloroethylene (TCE) as a human carcinogen, specifically citing "sufficient evidence in humans for the carcinogenicity of trichloroethylene. Trichloroethylene causes cancer of the kidney."[2] In addition, available evidence has provided a cohesive database supporting TCE as a known kidney carcinogen. This has been demonstrated in both human and animal studies, with mechanistic data suggesting that the carcinogenic effect of TCE results from its metabolism into genotoxic and cytotoxic intermediates that target the kidney and cause DNA strand breaks and mutations in tumor suppressor genes. The relationship between TCE exposure and kidney cancer risk has been documented in direct occupational exposure as well as residential chronic exposure at low to moderate doses. A study examining kidney cancer risk associated with historic groundwater contamination revealed the 50^{th}-75^{th} percentile of estimated exposure over a 15 year period was associated with an increased risk of kidney cancer with adjusted odds ratio (OR) of 1.78 95% confidence interval (CI) compared to $<50^{th}$ percentile.[3] In this study, the maximum measured groundwater TCE levels varied widely, with estimated TCE exposure levels generally ranging from 0-27.6 ug/L.[3]

Another study providing epidemiologic evidence supporting the association between TCE and renal cell carcinoma risk examined occupational TCE exposure in several European countries.[4] TCE exposure was categorized into one of three levels ranging from 0-<27ug/m^3, 27-270 ug/m^3 and >270 ug/m^3, with almost all TCE exposure occurring at least 20 years before disease onset.[4] For TCE exposure, ORs were significantly elevated for all exposure indices (OR = 1.63-2.34).[4] In addition, this study examined the association between TCE exposure and renal cell carcinoma risk after stratification by GSTT1 genotype, which revealed significant associations among subjects exposed to TCE with an active genotype (OR 1.88; 95% CI) but not among GSTT1 nulls (OR 0.93, 95% CI).[4] The findings of this study support the genotoxic mechanism believed to be causative in the development of renal cell carcinoma in these cases. A follow up analysis examined the association between TCE exposure and subtypes of clear cell renal cell carcinoma, with clear cell B subtypes demonstrating a statistically significant elevated measure of association (OR 3.09).[5]

Additional studies include Karami et al (2012) which also demonstrated that TCE can cause kidney cancer, as the authors performed a meta-analysis of 9 cohort studies which resulted in an overall elevated relative risk of 1.26 (95% CI 1.02-1.56) for TCE exposure and renal cancer.[6] Another meta-analysis included 23 studies: 16 cohort and 7 case-control.[7] This study demonstrated significantly elevated measures of association across all studies (RR 1.42), in only case-control studies (RR 1.33), and in only studies with well documented exposure assessment (RR 1.34).[7]

In addition to these references, there is literature directly relating to the toxins in the water at Camp Lejeune that supports the causal association between TCE and kidney cancer. Bove et. al. 2014a specifically studied the toxins in the water at Camp Lejeune and found associations between the Camp Lejeune water with all the chemicals at issue (TVOCs) and also individual chemicals.[8] Bove et. al. 2014a found a monotonic exposure response for TVOCs at Camp Lejeune relating to kidney cancer with RR of 1.42 (low exposures), 1.44 (medium exposures) and 1.54

(high exposures).[8] The supplemental tables in this study specifically detail HR for cumulative exposures to TCE for the individuals exposed at Camp Lejeune.[8] The HR for cumulative exposures to TCE were 1.54 (low exposures), 1.21 (medium exposures) and 1.52 (high exposures).[8]

There were additional causal relationships found between the toxic water at Camp Lejeune/TCE in the water at Camp Lejeune and kidney cancer. For example, Bove 2024 (both cancer incidence and cancer mortality) support a causal association for individuals exposed to the water at Camp Lejeune and kidney cancer.[9,10]

Finally, just recently, the EPA gave public notice of a final rule change completely banning TCE in the United States.[11] In the public notice of EPA's ban of TCE, the EPA and its spokespeople specifically listed the connection between TCE and kidney cancer as a reason for the need for the ban.[11] In its notice and rule, it cited Camp Lejeune's water contamination as an example of how TCE can cause cancers, including kidney cancer, at low levels.[11]

I have read the general causation report of experts Dr. Benjamin Hatten and Dr. Steven Bird. These expert reports detail an extensive review of the epidemiology, toxicology and mechanism of action of TCE and kidney cancer. These reports are consistent with my review of the literature and support my opinions in this case.

VIII. Kidney Cancer Risk Associated with PCE, VC and Benzene

The IARC has classified both vinyl chloride (VC) and benzene as known human carcinogens and PCE as "probably carcinogenic to humans."[2,12] Available epidemiologic data is consistent with toxicological evidence of PCE's carcinogenicity.

a. PCE

Mechanistically, PCE is thought to induce kidney cancer via genotoxicity, oxidative stress leading to DNA strand breaks and mutations, and direct cellular cytotoxicity. Epidemiologic studies involving PCE exposure demonstrate an association with kidney cancer. Aschengrau *et al.* reviewed the cancer risk experienced by a cohort of individuals exposed to PCE via contaminated water supplies on Cape Cod, Massachusetts.[13] Following this discovery, the Massachusetts Department of Health observed "elevations in cancer mortality" in affected areas.[13] This population was then matched to population-based controls to define the risk of cancers for the Cape Cod cohort.[13] The authors found that any PCE exposure (OR 1.23) and low PCE exposure (OR 1.36) demonstrated elevated measures of association with kidney cancer in an analysis not accounting for latency.[13]

The 2018 ATSDR Morbidity Study of Marines and civilians at Camp Lejeune found there was a monotonic exposure-response relationship between kidney cancer risk and TCE/ PCE exposure for Marines.[14] ORs were ≥1.5 for both TCE and PCE in Marines and for TCE/PCE in civilian employees.[14] In addition, an occupational case-control study published after the ATSDR Assessment reported an OR of 3.0 (95% CI: 0.99, 9.0) for kidney cancer among those with high PCE exposure intensity and high cumulative exposure after excluding those with ≥50% probability of TCE exposure.[15]

Many studies examining PCE exposure in occupations involve the dry-cleaning industry. For example, an elevated measure of association (SMR 1.41) for kidney cancer mortality was reported in a cohort study of dry

cleaner union members who worked in PCE exposed shops for at least a year prior to 1960 with up to a 20-year latency period.[16]

Further, the EPA just enacted a rule banning PCE products and in that rule used as a basis that PCE is causally associated with kidney cancer and that PCE can cause kidney cancer at low levels.

b. Vinyl Chloride

Mechanistically, vinyl chloride is thought to induce kidney cancer via oxidative stress leading to DNA strand breaks and mutations and the formation of DNA adducts. A DNA adduct is a segment of DNA that is chemically bonded to a cancer-causing chemical, inducing carcinogenesis.

There are epidemiologic studies involving vinyl chloride exposure that demonstrate an association with kidney cancer. Hu et el al (2002) demonstrated an increased risk of renal cell carcinoma in males with occupational exposure to vinyl chloride, in a dose-response manner, with the excess risk being significantly associated to duration of exposure.[17] Compared with no exposure to vinyl chloride, the adjusted OR was 2.0 (95% CI = 1.2–3.3).[17] In addition, Bove at al (2014a) found an elevated measure of association (HR 1.55) for kidney cancer deaths of military personnel stationed at Camp Lejeune compared to Camp Pendleton with at least low exposure to vinyl chloride.[8] Bove et al (2014a) found significantly increased HR at low, medium and high levels of exposure to VC; 1.66 (low exposure), 1.61 (medium exposure) and 1.51 (high exposure).[8]

c. Benzene

Mechanistically, benzene is thought to induce kidney cancer via its metabolites inducing oxidative stress leading to DNA strand breaks and mutations and the formation of DNA adducts.

There are epidemiologic studies involving benzene exposure that demonstrate an association with kidney cancer. The Hu study (2002) demonstrated an increased risk of renal cell carcinoma in males with occupational exposure to benzene, in a dose-response manner, with the excess risk being significantly associated to duration of exposure.[17] Compared with no exposure to the specific chemical, the adjusted OR was 1.8 (95% CI = 1.2–2.6).[17] Another occupational study of benzene exposure and kidney cancer was published by Greenland et al (1994).[18] This case-control study of benzene exposure in transformer manufacturing workers in Massachusetts found an OR of kidney cancer with benzene exposure of 4.29 (95% CI 1.33-13.8).[18] In addition, Seyyedsalehi et al (2024) performed a meta-analysis of 29 studies and found an association between occupational benzene exposure and kidney cancer, with an OR 1.20 (95% CI 1.03-1.39).[19]

I have read the general causation report of expert Dr. Benjamin Hatten and Dr. Steven Bird. These expert reports detail an extensive review of the epidemiology, toxicology and mechanism of action of PCE, VC and Benzene and kidney cancer. These reports are consistent with my review of the literature and support my opinions in this case.

IX. Impact of TCE, PCE, VC and Benzene Exposure from Camp Lejeune

The Agency for Toxic Substances and Disease Registry (ATSDR) has completed and reviewed several epidemiological studies and meta-analyses to determine if personnel and civilians were at increased risk for

certain health effects from exposure to this contaminated water.[1] The evidence from the methodological studies establishes that exposure to the levels of the toxins in the drinking water at Camp Lejeune are causes of kidney cancer.[1] All meta-analyses that evaluated epidemiological studies of high utility were based on reports from agencies mandated to evaluate the health risk of the chemicals, including the IARC (2014), EPA (2011) or NTP (2015).[2,20,21] Interpretation of the findings in meta-analyses published and reviewed in the scientific literature for TCE exposure and kidney cancer outline the magnitude of the adjusted Hazard Ratio (HR) between 1.2 and 1.4 across multiple studies, the precision of the effect estimates (CI>95%) and examine the impacts of unmeasured potential confounders and exposure misclassification on the HR estimate.[7,22] As noted, other studies in the literature have linked exposure to PCE, VC and benzene to the development of malignancies, including kidney cancer.

Based upon these studies and a literature review of occupational and environmental studies, the ATSDR report assessed the strength of the evidence supporting the causality of kidney cancer from TCE exposure.[1] The conclusion was that sufficient causal evidence exists linking TCE exposure and kidney cancer.[1] There was a monotonic exposure-response relationship between kidney cancer risk and TCE/ PCE exposure for Marines.[14]

There is additional epidemiologic literature relating specifically to the water at Camp Lejeune finding a causal relationship with kidney cancer, including Bove 2014a, Bove 2014b, the ATSDR 2018 mortality study, the 2024 Bove mortality study and the 2024 Bove cancer incidence study.[8,9,10,14,23]

X. The Levels of the Toxins in the Water at Camp Lejeune

ATSDR conducted historical reconstruction modeling to estimate the monthly average contaminant levels in the Tarawa Terrace (TT) and Hadnot Point (HP distribution systems.[1] Median estimates from the HP distribution system for TCE was 366ug/L (range 0-783ug/L), PCE 15ug/L (range 0 to 39ug/L) and VC 22ug/L (range 0 to 67ug/L), all of which exceed the EPA's listing of the maximum contaminant level (MCL) for the volatile organic compounds in drinking water in the United States.[1] These values are 5 ug/L for TCE, PCE and benzene; 2 µg/L for vinyl chloride.[1] In addition, the estimated drinking water concentrations of benzene consistently exceeded the current 5 ug/L MCL. This median estimate of TCE within the drinking water also exceeds median values observed to be associated with an increased risk of renal cell carcinoma in several epidemiologic studies referenced within this report.[1]

There are three known exposure pathways from contaminated water: ingestion, inhalation and dermal absorption. Each pathway contributes to level of chemicals within the body, their known biological effects, and therefore to the overall cancer risk.

In reviewing the General Causation Expert Report of Benjamin Hatten, M.D, M.P.H, Dr. Hatten states "Given that the exposure of interest is water contaminated with multiple culprit compounds, the body of literature that directly examines the Camp Lejeune population exposed to the contaminated water system best answers the question of what levels of exposure are associated with kidney cancer." I agree with this statement, and it supports my opinions in this matter as to the causal connection between the Camp Lejeune water and Mr. Howard's kidney cancer.

Exposures to TCE, PCE, benzene and vinyl chloride at Camp Lejeune occurred simultaneously. TCE and PCE are Camp Lejeune water contaminants with a sufficient body of evidence for causation of kidney cancer, with non-

monotonic exposure-relationships evident in studies involving Camp Lejeune.[8] Benzene and vinyl chloride are Camp Lejeune water contaminant with a body of evidence that meets the as likely as not standard for causation of kidney cancer. Therefore, an exposure to these compounds that is demonstrably hazardous to humans at Camp Lejeune and is causally associated with kidney cancer is the lowest cumulative exposure category that demonstrates an elevated measure of association.

The RR for the cumulative exposure of each individual chemical as it was causally related to kidney cancer were as follows:

PCE: 1.40 (low exposures), 1.82 (medium exposures) and 1.59 (high exposures)[8]

VC: 1.66 (low exposures), 1.61 (medium exposures) and 1.51 (high exposures)[8]

Benzene: 1.31 (low exposure), 1.38 (medium exposures) and 1.36 (high exposures)[8]

TCE: 1.54 (low exposure), 1.21 (medium exposures) and 1.52 (high exposures)[8]

Dr. Hatten also states "the most relevant evidence for on-base exposures is a monotonic exposure-response relationship with TVOC rather than any individual component exposure (Bove 2014a). Thus, the lowest exposure category to cumulative TVOC with a monotonic dose-response provides evidence of a low level of Camp Lejeune water that is hazardous to human health and a known cause of kidney cancer." I agree with this statement and Dr. Hatten's report supports my opinions in this matter.

In Bove (2014a) the classification for low, medium and high exposures were:

TVOCs: >1 – 4600 ug/L-months (low exposure), >4600 – 12,250 ug/L-months (medium exposures) and >12,250 - 64,016 ug/L-months (high exposure)[8]

TCE: >1 – 3,100 ug/L-months (low exposure), >3,100 – 7,700 ug/L-months (medium exposure), >7,700 – 39,745 ug/L-months (high exposure)[8]

PCE: >1 – 155 ug/L-months (low exposure), >155 – 380 ug/L-months (medium exposure), >380 – 8,585 ug/L-months (high exposure)[8]

Vinyl chloride: >1 – 205 ug/L-months (low exposures), >205 – 500 ug/L-months (medium exposures), >500 – 2,800 ug/L-months (high exposures)[8]

Benzene: 2 – 45 ug/L-months (low exposures), >45 – 110 ug/L-months (medium exposures) >110 - 601 ug/L-months (high exposures)[8]

Mr. Howard meets the criteria for the medium exposure categories for each of the individual chemicals and also TVOC exposure in this study.

The Camp Lejeune literature also analyzed exposure by time duration on base. A duration-based intensity of exposure is also supported by the Camp Lejeune literature with a monotonic exposure response evident.[10] The lowest duration category in the monotonic exposure-response finding that demonstrates an elevated measure of association is a level that is hazardous to human health and a known to cause kidney cancer. This is the "low" duration group with 1-5 quarters on base (HR 1.36).[10] Mr. Howard had similar exposure.

Dr. Hatten states in his report "To summarize, if an individual was present at Camp Lejeune and exposed to the levels of the chemicals above, this individual would have been exposed to levels of the water at Camp Lejeune that are hazardous to humans generally and are known to cause kidney cancer."

There were other levels shown in the literature that causally connect the toxins at issue in this case and kidney cancer. These were shown in the general causation reports for Drs. Hatten and Bird as well as cited elsewhere in this report. I will not repeat all these levels in this section, but all should be noted to be relevant to this analysis.

XI. Specific Causation: TCE, PCE, VC and Benzene Exposure and Allan Wayne Howard's Renal Cell Carcinoma

There are risk factors linked to an increased risk in the development of renal cell carcinoma. Those include exposure to the toxic chemicals noted above, tobacco use, prolonged hypertension, and excess body weight. An association between occupational risk factors and renal cell carcinoma has also been established in several epidemiologic studies.[6] Occupations that have been linked to renal cell carcinoma include the agricultural, dry cleaning and mechanical industries.

We employ scientific evidence, to attempt to ascertain whether exposure to the known carcinogens in the Camp Lejeune water were the cause of the Mr. Howard's kidney cancer. Based upon the review of Mr. Howard's medical records, his time stationed at Camp Lejeune, and review of the scientific and epidemiological evidence, it is my opinion that it is as likely than not that his exposure to the contaminated water at Camp Lejeune was the cause of his kidney cancer.

The following factors support my opinion:

(1) ATSDR historical reconstruction modeling to estimate the monthly average contaminant levels in the Tarawa Terrace (TT) and Hadnot Point (HP) distribution during the relevant times indicate that Mr. Howard was exposed to water with TCE, PCE, Vinyl chloride and Benzene contamination levels exceeding carcinogenic levels observed in epidemiologic studies demonstrating an increased risk of kidney cancer.[1]

(2) Allan Wayne Howard was stationed at the Mainside Barracks for 449 days from September 4, 1977 through February 8,1979 (not including time away from the base for annual leave and a deployment to the Caribbean from January 31 through March 7, 1978). This included time where he lived at the Mainside Barracks. The soldiers and civilian personnel at Camp Lejeune typically experienced multiple routes of exposure. In his deposition testimony, Mr. Howard stated that he continued to eat and hydrate on the base daily, as well as shower there a minimum of once per day for five to ten minutes. Scientific

2. [redacted]

3. [redacted]

4. The Government claims the exposure to the chemicals at Camp Lejeune may not have been sufficient to have caused Mr. Howard's kidney cancer. This argument lacks merit because of the discussion above with regards to the levels of his exposure to both the individual toxins as well as total volatile organic compounds that are hazardous to humans generally and are known to cause kidney cancer.

5. The Government claims that the length of time between exposure and diagnosis of kidney cancer may indicate an alternative cause of Mr. Howard's kidney cancer. This argument lacks merit because many studies referenced in this report utilized significant latency periods (10-20 years) to ensure that the exposure to the Camp Lejeune water system occurred sufficiently prior to the diagnosis of kidney cancer[8,23] Two of these studies even conducted sensitivity analyses with up to 20-year lags without substantive changes in results.[8,23] Analysis of these study designs using the Bradford Hill factors provides evidence for causation that accounts for the principle of temporality, referring to the principle that the exposure of interest must have occurred prior to the development of the disease process of interest to be a cause.

XV. Bradford Hill Factors

Multiple studies reviewed demonstrate an association between exposure to the contaminated Camp Lejeune water system and kidney cancer among Marines and civilians.[8,9,10,14,23] The Bradford Hill considerations are employed here for a structured analysis to determine whether this particular association with Mr. Howard is causal, and specifically, whether that it is as likely as not that this exposure was the cause of Mr. Howard's kidney cancer.

a. Strength of Association

Strength of association is demonstrated by statistical significance. Multiple studies discussed in this analysis demonstrate elevated measures of association between the Camp Lejeune water system that Alan Wayne Howard was exposed to and kidney cancer.[8,9,10,23]

b. Consistency

Consistency refers to studies being done in different populations yielding similar results. Multiple cohort[8,9,10,23] and case control[14] studies reached similar conclusions, providing consistent evidence between an association between exposure to the water system at Camp Lejeune and kidney cancer.

c. Exposure-Response

Studies referenced in this report have demonstrated a monotonic exposure-response relationship between increased TVOC exposure and duration at Camp Lejeune.[8,23] This was a consistent finding despite varied methods of determining exposure within these studies. Alan Wayne Howard, during his time at Camp Lejeune, was exposed to the levels of the chemicals listed above, and both his exposure levels to the individual toxins as well as total volatile organic compounds are hazardous to humans generally and are known to cause kidney cancer.

d. Temporality

Temporality refers to the principle that the exposure of interest must have occurred prior to the development of the disease process of interest to be a cause. Significant latency periods (10-20 years) were used in studies referenced in this report to ensure that the exposure to the Camp Lejeune water system occurred sufficiently prior the diagnosis of kidney cancer.[8,23] [redacted]

e. Biological Plausibility

This refers to the concept that a correlation between exposure and a disease process is causal based upon epidemiologic evidence. As discussed, TCE, PCE, vinyl chloride and benzene, all contaminants found in the water at Camp Lejeune, all meet the "as likely as not" standard for causation of kidney cancer. TCE and PCE have well documented mechanisms of kidney carcinogenesis, and vinyl chloride and benzene are both known carcinogens with biologically plausible mechanisms for causation of kidney cancer. The totality of the scientific evidence reviewed meets the biologic plausibility standard for Mr. Howard's exposure to the Camp Lejeune water and kidney cancer.

f. Analogy

Alan Wayne Howard's exposure to these toxins in the Camp Lejeune water system are analogous to other contaminated water systems that have been studied for association with kidney cancer, including two systems

referenced in this report.[3,13] In addition, there is ample evidence of occupational exposures involving TCE, PCE, vinyl chloride and benzene that provide analogous evidence of causation to kidney cancer.

g. Specificity

The consideration of specificity is limited given that fact that the contaminants in the Camp Lejeune water system are known to cause other adverse health outcomes, including cancer in other organs. In addition, there are other unmodifiable and modifiable known risk factors to kidney cancer. As stated, [redacted] and his only known exposure was to the contaminants in the Camp Lejeune water system.

h. Coherence

The contaminants in the Camp Lejeune water system are known carcinogens, and literature reviewed includes mechanistic, human and animal studies that provide coherent data demonstrating the association between exposure to the water at Camp Lejeune and the development of kidney cancer.

i. Summary

When the abundant scientific and epidemiologic evidence that directly examines the Camp Lejeune water exposure and the development of Mr. Howard's kidney cancer is considered through the Bradford Hill analysis, it is my conclusion that the exposure is more likely than not a cause of kidney cancer. Given Alan Wayne Howard's known exposure to the Camp Lejeune water system, the levels found at Camp Lejeune during the relevant time period, and his lack of other risk factors, it is more likely than not to be the cause of his kidney cancer. This analysis helps put weight behind the causal relationship between the water at Camp Lejeune and Mr. Howard's kidney cancer for purposes of the differential diagnosis and causal relationship.

XVI. Mr. Howard's Injuries

[redacted]